Segmental Portal Hypertension

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Isolated obstruction of the splenic vein leads to segmental portal hypertension, which is a rare form of extrahepatic portal hypertension, but it is important to diagnose, since it can be cured by splenectomy. In a review of the English literature, 209 patients with isolated splenic vein obstruction were found. Pancreatitis caused 65% of the cases and pancreatic neoplasms 18%, whereas the rest was caused by various other diseases. Seventy-two per cent of the patients bled from gastroesophageal varices, and most often the bleeding came from isolated gastric varices. The spleen was enlarged in 71% of the patients. A correct diagnosis in connection with the first episode of bleeding was made in only 49%; 22% were operated on because of gastrointestinal bleeding, but the cause of bleeding was not found. The diagnosis should be suspected in patients with gastroesophageal varices, but without signs of a liver disease, especially if isolated gastric varices are found. The diagnosis is confirmed by portography.

ATIENTS WITH EXTRAHEPATIC portal hypertension constitute 5-10% of all patients with portal hypertension (PH). 1-3 Isolated obstruction of the splenic vein leads to elevated pressure in the spleen and is a form of extrahepatic PH. The condition is present in only about 5% of patients with extrahepatic PH, 4.5 but it is important to diagnose it, since it is the only form of PH that is definitely curable. 6

Sutton et al.⁷ were able to find 54 cases in their review of the English literature between 1900 and 1968, and the condition is still so rarely diagnosed that only a few can gain experience with many patients. To outline recent years' experience with splenic vein obstruction, we have in the light of a topical case⁸ reviewed the literature of this form of extrahepatic PH, referred to as segmental,⁹ left-sided,¹⁰ regional,⁷ localized,¹¹ compartmental,¹² lienal,¹³ or splenoportal hypertension.¹⁴

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Materials and Methods

We have reviewed the English literature after Sutton et al., i.e., from 1969 to 1984. Defining isolated splenic vein obstruction as total or partial occlusion of the lumen of the splenic vein leading to formation of collaterals and with a normal portal and superior mesenteric vein, we found 73 references on this subject. Ten of the references were review articles and articles on diagnostics, in which the individual patients were not identified. 6,7,15-22 The remaining 63 references described a total of 209 patients with isolated splenic vein obstruction. 10-14,23-80 These patients are grouped according to sex, age, fundamental disease, symptoms, characteristic findings, diagnostics, treatment, and prognosis.

Results

Sex and Age

In the case of 131 patients, both sex and age were stated. There were 89 men (68%) with a median age of 48 years (ranging from 10 months to 85 years). The women had a median age of 45 years (ranging from 2 to 75 years).

Etiology

The cause of splenic vein obstruction was described in 187 patients (Table 1). In 65% of the cases, the cause was pancreatitis, and 33% of these patients had a pancreatic pseudocyst. Benign or malignant pancreatic tumors were the cause of splenic vein obstruction in 18% of the patients, whereas 17% of the cases were caused by a number of different diseases. In nine cases, isolated splenic vein obstruction was accompanied by cirrhosis of the liver, and, thus, a simultaneous presence of generalized and seg-

mental PH was seen in these patients. In addition to cirrhosis of the liver, four patients also had chronic pancreatitis, which was probably the original cause of the splenic vein obstruction. Two patients had splenic vein obstruction after the performance of portosystemic shunt. Only in three patients could no cause of splenic vein obstruction other than cirrhosis of the liver be found.

Symptoms

The patients' symptoms appear in Table 2. The most common symptom, which was present in 115 patients (72%), was bleeding from gastroesophageal varices. Usually, the bleeding was serious, in the form of hematemesis or melena. Often, there had been symptoms of a fundamental disease prior to or coinciding with the episode of bleeding. In 25 patients (16%), however, the bleeding was not preceded or followed by other symptoms, even though a cause of splenic vein obstruction could be demonstrated in 21 of these patients.

Abdominal pain without bleeding was present in 23%, often accompanied by other symptoms of chronic pancreatitis, a pancreatic pseudocyst or pancreatic cancer. One patient presented with encephalopathy without gastrointestinal bleeding.⁴³ The cerebral symptoms disappeared after splenectomy and the closure of a large collateral vessel from the splenic and superior mesenteric veins to the renal vein. Several liver biopsies, however, showed chronic persistent hepatitis, and, thus, this disease may have contributed considerably to the encephalopathy. Encephalopathy because of splenic vein obstruction was not demonstrated in any other patient.

Findings

A characteristic finding in the case of segmental PH is isolated gastric varices. The conditions in the esophagus and the stomach were described by means of angiography, upper endoscopy, and/or perioperative findings in 191 patients, 144 of whom (75%) had isolated gastric varices, whereas only 46 patients had varices in both the esophagus and the stomach. One patient had no gastroesophageal varices but did have varices in the left flexure of the colon.⁴⁴

Splenomegaly was present in 110 (71%) of the 154 patients whose spleens were described according to size. The basis on which to estimate the size of the spleen, however, varied a lot from one article to another. Itzchak and Glickman¹⁴ went thoroughly into this subject of the size of the spleen in connection with isolated splenic vein obstruction. On the basis of arteriographical findings, they found that the spleen was enlarged in eight (42%) of 19 patients. Only in a few patients did the enlarged spleen

TABLE 1. The Cause of Isolated Splenic Vein Obstruction in 209 Patients

Diagnosis		N
Pancreatitis, total		122
Chronic pancreatitis without pseudocyst	56	
Acute pancreatitis without pseudocyst	5	
Pancreatic pseudocyst	40	
Pancreatitis, unspecified as to acute and		
chronic inflammation and pseudocyst	21	
Pancreatic neoplasms, total		34
Adenocarcinoma	23	
Cystadenoma	5	
Islet cell tumor	6	
Idiopathic		6
Cirrhosis of the liver		3
Consequences of umbilical vein catheter		3
Retroperitoneal lymphoma		3
Retroperitoneal fibrosis		3 3 3 2
Adenocarcinoma in the kidney		2
Consequences of surgical portosystemic shunt		2 2
Consequences of resection of the stomach		2
Various causes*		8
Cause not stated		22
Total		209

^{*} One of each of the following: congenital pancreatic cyst, retroperitoneal abscess, wandering spleen, gastric ulcer, abdominal trauma, hydatid cyst in the spleen, cavernous splenic vein, sarcoma in the colon.

produce symptoms because of size or leukocytopenia and thrombocytopenia.

Diagnostics

Often a long period of time had passed from the appearance of the first symptoms till the diagnosis was made. Thus, the period of time, from the first episode of bleeding till the diagnosis was made, was stated in 82 patients. Only 49% had their diagnosis made in connection with the first episode of bleeding, whereas 1 month to 12 years (median: 11 months) passed before the correct cause of the bleeding was found in the remaining patients. Twenty-

TABLE 2. Symptoms in 209 Patients with Isolated Splenic Vein Obstruction

Symptom		N	
Visible bleeding, total		99	
Visible bleeding and other, previous symptoms	74		
Visible bleeding without other symptoms	25		
Occult bleeding and other, previous symptoms		16	
Abdominal pain without bleeding, possibly			
accompanied by other symptoms		36	
Other symptoms*		9	
Symptoms not stated		49	
Total		209	

^{*} Three splenomegaly, 2 icterus, 1 encephalopathy, 1 thrombocytopenia, 1 splenic rupture, 1 diabetes mellitus.



FIG. 1. Splenoportography in a 15-year-old boy with idiopathic isolated splenic vein obstruction and bleeding from gastric varices (V). The normal portal vein (P) is filled by the gastric varices and by the prominent gastroepiploic veins (G). There are no esophageal varices.

six (22%) of the 116 patients who bled from the gastrointestinal tract were operated on for the bleeding, but the cause of the bleeding was not demonstrated at the operation. The correct diagnosis was not made until new episodes of bleeding and new examinations had taken place.

Treatment

Most patients were treated by splenectomy. The course following splenectomy was stated in the case of 72 patients

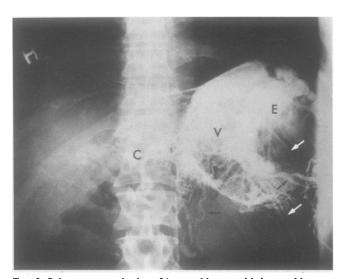


FIG. 2. Splenoportography in a 54-year-old man with inoperable pancreatic cancer. There are prominent splenosystemic collaterals to the retroperitoneum (black arrows) and intercostal veins (white arrows). There is some extravasation of the contrast (E). There had been no gastrointestinal bleeding, and the varices (V) in the fundus and the coronary vein (C) are small. There are no esophageal varices.

with a median period of observation of 12 months (ranging from 0 months to 24 years). Only two of these patients had bleeding following splenectomy. 70,73 One bled because of cirrhosis of the liver with generalized PH and coagulation disorders. The other patient did not stop bleeding until total gastrectomy had been performed. Liver biopsy showed stasis, but no cirrhosis, and the cause of the rebleeding was not clear.

Thirteen patients had bleeding, but splenectomy was not performed. Four of these patients died shortly after the diagnosis was made because of persistent or recurrent bleeding and because of the fundamental disease. One of these patients was treated by mesocaval shunt, which did not affect the bleeding. ⁵³ None of the remaining nine patients bled again, but five died shortly after from the fundamental disease, and the other four were observed for only 6–12 months.

Splenectomy was not performed in only six patients without bleeding at the time of the diagnosis. ^{12,25,50,57} One of these patients bled 3 months later and was treated with percutaneous splenic embolization. ²⁵ None of the others bled, but the period of observation was only 8–18 months.

Discussion

Pathology

Obstruction of the splenic vein causes the pressure in the spleen to rise, and a number of splenoportal collateral systems will develop (Fig. 1), returning the blood from the spleen, bypassing the splenic vein, into the portal vein. 14,22,65 There is retrograde flow through the short gastric veins into submucous and subsequently varicose veins in the fundus and the cardia area, from where the blood flows hepatopetally through the coronary vein to the portal vein. Retrograde flow through the left gastroepiploic vein into the right gastroepiploic vein and the superior mesenteric vein also occurs. Often the gastroepiploic veins are connected with dilated veins in the omentum. The left gastroepiploic vein may also lead the blood into the left colic vein and the inferior mesenteric vein.

Splenosystemic collaterals may also be formed, such as retrograde flow through the short gastric veins into the submucous and subsequently varicose veins of the esophagus. Collaterals from the spleen to the retroperitoneum, renal vein, and intercostal veins are not so common (Fig. 2).

Cause

Splenic vein obstruction may be caused by compression of the vein by other organs or by thrombosis of the vein.⁵⁰ Often a combination of these two factors will be present.

In their review of the literature, Sutton et al. found that 35% of the cases of isolated splenic vein obstruction

were caused by tumors and 17% by pancreatitis. We found that tumors caused 21% and pancreatitis 65% of the cases of splenic vein obstruction. These differences may be due to an increase in the number of cases of pancreatitis and to an increase in diagnostic and especially angiographic activities during recent years.

The distribution on sex and age found in our review agrees with pancreatic diseases as the predominant cause of splenic vein obstruction.

Frequency

Segmental PH has been found in 4-6% of patients with extrahepatic PH.^{4,5}

In patients with chronic pancreatitis, segmental PH has been found in 5-37%, based on angiographic and perioperative findings. ^{9,81-83} The clinically and histologically most severe cases of chronic pancreatitis lead to the most severe changes in the splenic vein. ^{9,81} This is most likely the explanation of the different statements of the frequency of segmental PH, as the indications for performing portography and operation vary in the various materials. Complications of pancreatitis in the form of a pseudocyst seem to increase the frequency of splenic vein obstruction; Rösch found that 14 of 16 patients (88%) with pancreatic pseudocyst had splenic vein obstruction. ⁸¹

Symptoms and Findings

The symptoms and findings consist partly of those that can be traced back to the fundamental disease, such as pain, weight loss, or an abdominal mass, and partly of those caused by the segmental PH: gastrointestinal varices, probably with bleeding, and splenomegaly, which may be a hampering mass or which may give leukocyto- or thrombocytopenia.

Like others, ^{12,23,38} we found in our review of the literature that hematemesis and melena caused by bleeding from isolated gastric varices were the most important signs of isolated splenic vein obstruction. Segmental PH does not always produce symptoms, but the indications of the frequency of symptoms vary a lot in the literature. Thus, bleeding has been found in 12–69% of patients with segmental PH caused by pancreatitis. ^{37,82,84}

Diagnostics

A decisive point when diagnosing segmental PH is the demonstration of gastroesophageal varices in a patient who shows no signs of a liver disease. Especially in the case of isolated varices in the stomach, isolated splenic vein obstruction should be suspected. It may be difficult by endoscopy to distinguish the varices from normal mucosal folds, and the varices are only demonstrated by means of endoscopy in 0-33% of the patients. 14,37,38 In

the case of barium examination of the stomach with double contrast technique, the varices are visualized in about 80% of the patients.^{38,45}

The diagnosis can be confirmed by portography. Recent works recommend the performance of arterioportography or indirect portography. 18,76 The characteristic finding on injection of contrast in the celiac artery is that in the venous phase the splenic vein is nonvisualized, whereas collaterals between the spleen and the portal vein are visualized.^{22,76} Injection in the superior mesenteric artery shows in the venous phase a normal portal vein with the coronary vein nonvisualized. An improved visualization of the conditions in the area of the splenic vein is obtained by selective injection of contrast medium in the splenic artery.¹⁸ In spite of this, the contrast in the portal vessels is not so dense as in the case of direct portographies: splenoportography and percutaneous transhepatic portography, which also improve the demonstration of the collaterals.⁸⁵ Splenoportography provides better visualization of the splenic vein than does percutaneous transhepatic portography, and therefore some surgeons prefer splenoportography as the first method of examination when splenic vein obstruction is suspected.¹⁷ Another advantage of this examination is the ability to measure the splenic pulp pressure. Percutaneous transhepatic portography^{34,86} and ultrasound scanning^{21,87} have so far been used to demonstrate only a few cases of splenic vein thrombosis.

A characteristic operative finding is dilated, tortuous vessels around the spleen and stomach; the gastroepiploic vein, especially, is marked. The spleen is often enlarged, the liver is normal, and there are no dilated vessels in the right side of the abdomen. Finally, it may be possible to demonstrate the fundamental cause of the splenic vein obstruction. The diagnosis can be confirmed by perioperative splenoportography and pressure measurement, if portography has not been performed before operation. Est

Treatment and Prognosis

Segmental PH is treated by splenectomy. Thus, the flow to the varices and the other collaterals is minimized, and the pressure in the collaterals is reduced. To reduce the perioperative loss of blood, a ligature of the splenic artery is recommended before mobilizing the spleen with the great number of collaterals in the ligaments. ^{12,33} During the operation, further intervention can be made because of the fundamental disease. In the case of a pancreatic pseudocyst, some surgeons ^{12,33} prefer cystojejunostomy to cystogastrostomy in order to reduce the risk of bleeding from the gastric mucosa of the stomy.

In two patients, percutaneous embolization of the splenic artery was performed. One patient was observed for 2 years without bleeding.³⁴ The other developed a large abscess of the spleen.²⁵

The question of prophylactic splenectomy, *i.e.*, splenectomy because of segmental PH in the absence of gastrointestinal bleeding, is not sufficiently clarified. ^{12,46,65} In our review, we found only six patients in whom prophylactic splenectomy was not performed, and the course of these patients does not enable us to draw any reliable conclusions. One argument in favor of not performing splenectomy is a few instances of regression of the splenic vein obstruction in connection with improvement of the fundamental disease. ^{31,50,81}

Conclusion

Isolated splenic vein obstruction is a rare but important condition that gives rise to the formation of gastrointestinal varices, which, in their turn, imply a risk of serious upper gastrointestinal bleeding. The condition should be considered, especially if isolated gastric varices are present in a patient who shows signs of a pancreatic disease and no signs of a liver disease. The diagnosis can be confirmed by arterioportography, probably accompanied by spleno-portography.

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References

- 1. Burcharth F. Percutaneous transhepatic portography. I. Technique and application. AJR 1979; 132:177-182.
- Hamilton DW, Hunt AH. Extrahepatic portal obstruction. Med J Aust 1970; 1:493-499.
- Novis BH, Duys P, Barbezat GO, et al. Fibreoptic endoscopy and the use of the Sengstaken tube in acute gastrointestinal haemorrhage in patients with portal hypertension and varices. Gut 1976; 17:258-263.
- Grünert RD. Pathogenese und Klassifikation der Erkrankungen mit Pfortaderhochdruck. Heidelberg: Alfred Hüthig, 1967.
- Belli L, Puttini M, Marni A. Extrahepatic portal obstruction: clinical experience and surgical treatment in 105 patients. J Cardiovasc Surg 1980; 21:439-448.
- Babb RR. Splenic vein obstruction: a curable cause of variceal bleeding. Am J Dig Dis 1976; 21:512–513.
- Sutton JP, Yarborough DY, Richards JT. Isolated splenic vein occlusion. Arch Surg 1970; 100:623-626.
- Madsen MS, Sommer H, Petersen TH, et al. Variceal bleeding caused by segmental portal hypertension in association with situs inversus and malrotation. Annales Chirurgiae et Gynaecologiae 1985; 74: 185-187.
- Leger L, Lenriot JP, Lemaigre G. L'hypertension et la stase portales segmentaires dans les pancréatites chronique: a propos de 126 cas examinés par spléno-portographie et spléno-manométrie. J Chir (Paris) 1968; 95:599-608.
- Turrill FL, Mikkelsen WP. "Sinistral" (left-sided) extrahepatic portal hypertension. Arch Surg 1969; 99:365-368.
- Lavender S, Lloyd-Davis RW, Thomas ML. Retroperitoneal fibrosis causing localized portal hypertension. Br Med J 1970; 3:627– 628
- Salam AA, Warren WD, Tyras DH. Splenic vein thrombosis: a diagnosable and curable form of portal hypertension. Surgery 1973; 74:961-972.
- Rösch W. Isolated gastric varices: a hint of pancreatic disorders. Endoscopy 1974; 6:217-220.

- Itzchak Y, Glickman MG. Splenic vein thrombosis in patients with a normal size spleen. Invest Radiol 1977; 12:158-163.
- 15. Zannini G, Masciariello S, Pagano G, et al. Prehepatic portal hypertension: experience with 88 cases. Int Surg 1982; 67:311–315.
- Lane FC. Acute pancreatitis: at what point should you consider surgery? Med Times 1980; 108:38-43.
- Burcharth F, Nielbo N, Andersen B. Percutaneous transhepatic portography. II. Comparison with splenoportography in portal hypertension. AJR 1979; 132:183-185.
- 18. Herlinger H. Arterioportography. Clin Radiol 1978; 29:255-275.
- Itzchak Y, Glickman MG, Gottschalk A, et al. Hemodynamic and morphologic evaluation of the spleen after splenic vein ligation in the dog. Invest Radiol 1978; 13:155-160.
- Itzchak Y, Glickman MG. Duodenal varices in extrahepatic portal obstruction. Radiology 1977; 124:619-624.
- Webb LJ, Berger LA, Sherlock S. Grey-scale ultrasonography of portal vein. Lancet 1977; 2:675-677.
- Ruzicka FF, Rossi P. Arterial portography: patterns of venous flow. Radiology 1969; 92:777-787.
- Røder OC. Splenic vein thrombosis with bleeding gastroesophageal varices: reports of two splenectomized cases and review of the literature. Acta Chir Scand 1984; 150:265-268.
- Goldberg S, Katz S, Naidich J, Waye J. Isolated gastric varices due to spontaneous splenic vein thrombosis. Am J Gastroenterol 1984; 79:304–307.
- Jones KB, Koos PTD. Postembolization splenic abscess in a patient with pancreatitis and splenic vein thrombosis. South Med J 1984; 77:390-393.
- Bok EJ, Cho KJ, Williams DM, et al. Venous involvement in islet cell tumours of the pancreas. AJR 1984; 142:319-322.
- Porter BA, Grey CF, Link DP, et al. Splenic embolization monitored by the video dilution technique. AJR 1983; 141:1063-1065.
- Dewar G. Regional portal hypertension. S Afr J Surg 1983; 21:69–71.
- Bunt TJ, Hackler MT, Greene FL. Isolated splenic vein thrombosis: a curable variceal hemorrhage. South Med J 1983; 76:936-938.
- Harnar T, Johansen K, Haskey R, Barker E. Left-sided portal hypertension from pancreatic pseudotumor. Am J Gastroenterol 1982; 77:639-641.
- Hasan A, Ahmed M. Isolated splenic vein occlusion: a report of two cases. Journal of the Pakistan Medical Association 1982; 32:79– 80.
- Cummings R, Raju S. Splenic vein thrombosis: a curable form of portal hypertension. J Miss State Med Assoc 1982; 23:137–138.
- Keith RG, Mustard RA, Saibil EA. Gastric variceal bleeding due to occlusion of splenic vein in pancreatic disease. Can J Surg 1982; 25:301-304.
- Alwmark A, Gullstrand P, Ihse I, et al. Regional portal hypertension in chronic pancreatitis. Acta Chir Scand 1981; 147:155-157.
- 35. Koehler RE. Case 2. Gastrointest Radiol 1981; 6:365-370.
- Manenti A. Splenic vein obstruction secondary to pancreatic carcinoma. Acta Chir Belg 1981; 80:245-248.
- Little AG, Moossa AR. Gastrointestinal hemorrhage from left-sided portal hypertension: an unappreciated complication of pancreatitis. Am J Surg 1981; 141:153-158.
- Muhletaler C, Gerlock AJ, Goncharenko V, et al. Gastric varices secondary to splenic vein occlusion: radiographic diagnosis and clinical significance. Radiology 1979; 132:593-598.
- Aron R, Zapolanski A, Cooperman AM, Hermann RE. Regional portal hypertension. Cleve Clin Q 1979; 46:1-5.
- Law DK, Moore EE. Compartmentalized gastrosplenic and mesenteric venous hypertension after distal splenorenal shunt occlusion: response to mesocaval shunt and splenectomy. Surgery 1979; 85:579-582.
- Salam AA, Goldman M, Smith D, Hill HL. Gastric, intestinal, and gallbladder varices: hemodynamic and therapeutic considerations. South Med J 1979; 72:402-408.
- Fitzer PM, Stephens JR. Splenic vein obstruction with bleeding gastric varices: a diagnostic approach. Virginia Medical 1978; 105:850– 853
- Honda Y, Ueda M, Kyoi M, et al. An unusual case of portasystemic encephalopathy caused by splenic vein occlusion following gastrectomy. Am J Gastroenterol 1978; 69:590-593.

- Burbige EJ, Tarder G, Carson S, et al. Colonic varices: a complication of pancreatitis with splenic vein thrombosis. Am J Dig Dis 1978; 23:752-755.
- Cho KJ, Martel W. Recognition of splenic vein occlusion. AJR 1978; 131:439-443.
- Wilson AJ. Isolated splenic vein thrombosis: report of a single case and review of the literature. Br J Clin Pract 1978; 32:27-29.
- Lynch CT, Maunz DL. Gastroduodenal varices secondary to splenic vein thrombosis. J Maine Med Assoc 1977; 68:455–458.
- Amarnani NH, Broor SL, Varma RR, et al. Splenic vein thrombosis: a cause of gastric varices and upper gastrointestinal bleeding. Gastrointestinal Endoscopy 1977; 24:83–85.
- Haff RC, Page CP, Andrassy RJ, Buckley CJ. Splenectomy: its place in operations for inflammatory disease of the pancreas. Am J Surg 1977; 134:555-557.
- Marshall JP, Smith PD, Hoyumpa AM. Gastric varices: problem in diagnosis. Am J Dig Dis 1977; 22:947-955.
- Khan AH, O'Reilly CJ, Avakian VA, Lucina PA. Splenic vein thrombosis: an unusual case of gastric bleeding. Angiology 1977; 28:725-727.
- Wolf JH, Long RJ, Miller FJ, Jeffries GH. Pancreatic islet cell tumor presenting as bleeding gastric varices secondary to splenic vein occlusion. Am J Dig Dis 1977; 22:652-655.
- Larmi TKI, Mokka REM, Kairaluoma MI, et al. Gastric bleeding due to segmental portal hypertension: a case report. Acta Chir Scand 1976; 142:609-610.
- Hazenberg HJA, Gips CH. ¹³N-ammonia test and urea index in a patient with splenic vein obstruction and cirrhosis of the liver. Neth J Med 1976; 19:127-134.
- Scott RL, Himmelfarb EH. X-ray of the month. J Tenn Med Assoc 1976; 69:197–198.
- Codd JE. Splenic vein thrombosis: case report. Mo Med 1976; 73: 22-24.
- Wexler MJ, MacLean LD. Massive spontaneous portal-systemic shunting without varices. Arch Surg 1975; 110:995-1003.
- 58. Byerly WG. Thrombosis of the splenic vein with secondary rupture of the spleen. N Ca Med J 1975; 36:352-354.
- Wolloch Y, Chaimoff C, Lubin E, Dintsman M. Splenic vein thrombosis, segmental portal hypertension and bleeding esophageal varices produced by a congenital pancreatic cyst. Israel J Med Sci 1974; 10:670-673.
- Vos LJM, Potocky V, Bröker FHL, et al. Splenic vein thrombosis with oesophageal varices: a late complication of umbilical vein catheterization. Ann Surg 1974; 180:152-156.
- Smith SJ, Wilson JHP. Splenic vein obstruction. Neth J Med 1974; 17:54-57.
- Richman LS, Boehnke M. Splenic vein thrombosis: case report. Angiology 1974; 25:300-303.
- Gallardo-Navarra V, Phillips E, Neyfeld P, Sakwa S. Segmental portal hypertension and islet cell adenoma of the pancreas. JAMA 1973; 226:1466.
- Negus D, Cotton P. Splenic vein thrombosis with pancreatic pseudocyst. Proc Roy Soc Med 1973; 66:649–650.
- Johnston FR, Myers RT. Etiologic factors and consequences of splenic vein obstruction. Ann Surg 1973; 177:736-739.

- Goldstein GB. Splenic vein thrombosis causing gastric varices and bleeding. Am J Gastroenterol 1972; 58:319-325.
- Cope JR, Clendinnen DG, Jacquet N. Malignant splenic vein obstruction. Br J Radiol 1972; 45:855-857.
- Warshaw AL, Chesney TM, Evans GW, McCarthy HF. Intrasplenic dissection by pancreatic pseudocyst. N Eng J Med 1972; 287:72– 75.
- Ben-Ari G, Mitty H, Kark AE, Rudick J. Segmental portal hypertension due to congenital cavernous malformation of splenic vein. Mt Sinai J Med 1972; 39:271-278.
- Yale CE, Crummy AB. Splenic vein thrombosis and bleeding esophageal varices. JAMA 1971; 217:317-320.
- Sheers R. A pancreatic cystadenoma complicated by varices: case report. Br J Surg 1980; 67:144-145.
- Probst P, Rysavy JA, Amplatz K. Improved safety of splenoportography by plugging of the needle tract. AJR 1978; 131:445-449.
- Stone RT, Wilson SE, Passaro E. Gastric portal hypertension. Am J Surg 1978; 136:73-79.
- Cosgrove H, Legge D, O'Connell FX, Weir D. Angiographic evaluation of gastrointestinal haemorrhage complicating pancreatic disease. Clin Radiol 1978; 29:289-293.
- Rice RP, Thompson WM, Kelvin FM, et al. Gastric varices without esophageal varices: an important preendoscopic diagnosis. JAMA 1977; 237:1976–1979.
- Rousselot LM, Burchell AR. Splenic and arterial portography and hemodynamics in portal hypertension. *In Schiff L*, ed. Diseases of the Liver, 4th ed. Philadelphia: JB Lippincott, 1975; 368-423.
- Smulewicz JJ, Clemett AR. Torsion of the wandering spleen. Am J Dig Dis 1975; 20:274-279.
- Maclean N, Falconer CWA, Gilmour IEW, Webb JN. Islet cell tumours of the pancreas with portal varices and gastrointestinal haemorrhage. J R Coll Surg Edinburgh 1970; 15:206-212.
- Longstreth GF, Newcomer AD, Green PA. Extrahepatic portal hypertension caused by chronic pancreatitis. Ann Int Med 1971; 75:903-908.
- Looby WE, Bennett JR, Rehman A. Splenic hypertension in pancreatitis. Rocky Mountain Med J 1969; 66:29-32.
- Rösch J. Die Splenoportographie in der Diagnostik der Pankreaserkrankungen. Der Radiologe 1965; 5:274–281.
- Hess W. Die chronische Pankreatitis: Klinik, Diagnostik und chirurgische Therapie der chronischen Pankreopathien. Bern: H Huber, 1969.
- Tylén U, Arnesjö B. Angiographic diagnosis of inflammatory disease of the pancreas. Acta Radiol Diagn 1973; 14:215-240.
- 84. Rignault D, Mine J, Moine D. Splenoportographic changes in chronic pancreatitis. Surgery 1968; 63:571-575.
- Smith-Laing G, Camilo ME, Dick R, Sherlock S. Percutaneous transhepatic portography in the assessment of portal hypertension: clinical correlations and comparison of radiographic techniques. Gastroenterology 1980; 78:197-205.
- Wiechel KL. Direct selective catheterization of the portal venous system. Scand J Gastroenterol 1979; 14(suppl 53):123-130.
- Jeanty P, Brion JP, Gossum AV, Struyen J. Portal and splenic vein thrombosis: ultrasonic demonstration. J Belge Radiol 1982; 65: 45-47.